Progressive neurologic disease related to the brachial neuropathy does not develop. Recovery in patients with upper plexus involvement is more rapid than in patients with lower plexus involvement. A third of patients will have recovered at the end of the first year, two thirds by the end of the second year and approximately 90 percent by the end of the third year. Residual weakness is usually in shoulder girdle muscles. No specific therapy is available and use of steroids does not appear to be of benefit. Pain relief during the acute phase and physical therapy for rehabilitation of patients are useful.

The diagnosis of brachial plexus neuropathy can be made readily in patients who present with the typical pattern of acute pain in or around the shoulder followed by weakness and without other symptoms or signs indicative of root or spinal cord disease. When the onset is in the first two decades and if there is evidence of recurrent attacks, the genetic form of this neuropathy probably is present. The long-term prognosis is good and supportive therapy is sufficient.

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Treatment of Asymptomatic Carotid Bruit

ALTHOUGH it is not common practice, some physicians use angiography in all patients with carotid bruits and, if a surgically correctable lesion is found, carry out carotid endarterectomy. Javid and co-workers have reported on the outcome in 56 patients with asymptomatic carotid bruits in whom endarterectomy was done. One patient died during the operation, postoperative strokes developed in two and strokes months after the operation developed in two. However, none of the 15 patients who died during an average follow-up of 36 months died from stroke. Twelve died from acute myocardial infarctions, one from gastric hemorrhage, one from liver failure and one from suicide. Thompson and Patman compared the long-term outcome in 49 patients with asymptomatic carotid bruits who were operated on with 59 patients who were not. In four of the patients in the operated group strokes developed immediately following operation or months later. In the nonoperated group, strokes developed in 14 patients. Unfortunately, the experimental group and the control group were distinctly different and comparisons between the two are of doubtful value. The likelihood of the patient with an asymptomatic carotid bruit developing a stroke has not been adequately assessed. Recently, Fields and Lemak reported that only 15 percent of 79 patients with transient ischemic attacks in the internal carotid perfusion territory suffered from strokes causing disability or death.

In an asymptomatic patient with a carotid bruit (regardless of its hemodynamic significance) but with cholesterol emboli in the fundus, angiography should be done. Angiography should also be done in patients in whom unequivocal abnormalities are found on Doppler ultrasonography, ocular plethysmography and ophthalmodynamometry indicating decreased internal carotid blood flow. During an angiography the intracranial vasculature always must be visualized. Whether or not further studies should be done in other patients who do not fulfill the above criteria is uncertain. The morbidity and mortality associated with carotid endarterectomy should be weighted against the low but significant risk of cerebral infarction without operation. Today an acceptable surgical mortality for carotid endarterectomy is 1 percent or less.

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Use of Anticoagulants in the Treatment of Stroke

BEFORE CARRYING OUT anticoagulation in a patient with stroke, one must decide whether the stroke is due to ischemia or hemorrhage. The distinction can be made easily when a patient presents with the usual clinical signs of subarachnoid hemorrhage, which are severe headache and stiff